of a transformation of neutrophiles into eosinophiles. We were unable to procure a sufficient quantity of muscle to confirm or refute Brown's observation, that the change takes place in the muscular tissue.

It is unfortunate that our cases could not be kept under observation longer, in order to observe the duration of the eosinophilia. The only two observations on this point that we have been able to discover are Brown's third case, where the blood was normal five months after the patient's discharge with an eosinophilia of 34.7 per cent., and Stump's case, where there was still an eosinophilia of 15 per cent. after four and one-half months.

As a result of the study of this outbreak we would conclude:

1. That in some epidemics of trichinosis one or more of the most characteristic symptoms may be lacking in the majority of the patients.

2. That in these cases the examination of the blood is of the greatest value, as pathognomonic changes are found in it, consisting in (*a*) an eosinophile leucocytosis, (*b*) a relative decrease in the neutrophiles, and (*c*) in many, but not all, cases a decrease in the small mononuclear leucocytes early in the disease.

THE RELATION OF MIGRAINE TO EPILEPSY.[1]

WITH THE REPORT OF ILLUSTRATIVE CASES.

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The relation of migraine to epilepsy is one of much interest, partly on account of the prevalence of both diseases. Two cases which have recently come under my observation seem of sufficient importance to be reported, and offer a modest contribution to our knowledge of the subject:

Case I.—A. A. D., white, aged fifty-one years, a carpenter by trade, was referred to my clinic at the Polyclinic Hospital by Dr. S. Solis-Cohen, and to him I am indebted for the opportunity of studying the case.

Family History. Father died at the age of sixty-one years from "phthisis or spinal disease." Mother is living and well. One brother is living and well. The patient has had no children, but his wife had one miscarriage at the fourth month.

Previous History. The man had measles when a child, but his mother states positively that he had no convulsions in childhood. He denies having had venereal disease. He had "camp fever" in 1864, and was in bed for two weeks. He had some kidney trouble for two or three weeks in 1898, and at present he has to get up at night to pass his urine. His urine, however, is normal; it contains no casts, no albumin, and no sugar. He has used alcohol since he was eight years of age, but says he never drank to excess. He used tobacco to excess both in chewing and smoking, and about thirty years ago he smoked as many as thirty cigars daily; at present he smokes very little.

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[1] Read before the Philadelphia Neurological Society, October 23, 1899.

Seven years ago he first noticed numbness on the end of his tongue, on the right side, lasting a minute or two. He was unable to speak while this numbness existed, although he knew what he wished to say. The numbness may possibly have extended a little to the left side of the tongue. The attacks of numbness in the tongue were at first very infrequent, occurring only once or twice in a year. He believed they were due to the use of tobacco, and after he stopped chewing tobacco he thought the attacks were not so severe, but they still continued.

Two or three years ago—*i. e.*, four or five years after the attacks of numbness in the tongue began—he noticed the first attack of numbness in the right hand and arm. When he had one of these attacks he could not grasp an object with the fingers of the right hand, and the article slipped from his hand. He had a sensation in the right arm, and especially in the hand, which he described as "pins and needles." The numbness appeared in the right lower limb shortly after it was first noticed in the upper limb. When the hand and leg became affected the numbness was associated with a "drawing feeling," probably a sensation of tension. The numbness would begin in either extremity of the right side or in the face on the right side. At times when it began in the right upper limb it would descend to the right lower limb; at other times it would begin in the right upper limb and pass to the head; at other times it would begin in the right lower limb, pass to the right upper limb, and ascend to the head; but in the attacks in which he fell the numbness always, or nearly always, began in the back of the right hand and passed to the right side of the head; the right eye then *felt* as though it were drawn up and to the right and backward, and the right corner of the mouth upward. When he felt that he was in danger of falling he made grasping movements with his arms, and these, he believes, were made to secure support and prevent himself from falling. A man who observed him in one or two of his attacks says his face and eyes were *not* drawn upward. The patient has lost consciousness in his major attacks, usually after falling, and has remained unconscious about a half to three-quarters of an hour. No relaxation of the sphincters has occurred during an attack. He has not had any jerkings or twitchings, but a tremor of the hands has been observed during a major attack. A glass of water has frequently aborted an attack. When one of his major attacks is coming on he utters a sound like "Uh-uh-uh!" After an attack of unconsciousness is over he gets up and goes to work, although he feels bewildered. In his lighter attacks, lasting not over a minute, he has numbness of the right side, aphasia, and loss of power in the right hand, but is not unconscious. He has had only six major attacks—*i. e.*, attacks with unconsciousness—and all these have occurred within the last two years. In two of these attacks the tongue was injured. He feels so unusually well for twenty minutes or so before one of his major attacks begins that he knows he is about to have an attack, and can tell the approach of an attack by a drowsy feeling. His wife saw him in a severe attack, and she says he came into the room making gestures for her to call the neighbors. He did not speak, and his face seemed puffed out. He permitted himself to be laid on the floor, and soon lost consciousness, but had no convulsions. He has not been a sufferer from headache, nor has he at any time had visual disturbances—hemianopsia, scintillating scotoma, etc. He is seldom dizzy.

His knee-jerks are very active and equal. He has no objective sensory disturbances. His taste is normal. Sense of position in the extremities is normal except that a slight disturbance may exist in the right fingers. No indications of hysteria are detectable. He reads easily, and is not at all deaf to words or sounds. He writes without difficulty.[1]

The report of his ocular condition by Dr. H. F. Hansell is: "Cutting down in the anterior downward portion of the form field. Vision is good. Pupils are equal and react fairly to light and in convergence. The muscles are normal, and the optic nerves show slight effects of eye-strain, which is not of any especial importance."

The second case I was invited to see by Dr. S. M. Hamill:

CASE II.—A. B., twenty-one years of age, is a well-developed woman. The mother of the patient had attacks of tingling in both hands, disturbance of speech, with severe bilateral headache, but without visual disturbances, between her eighteenth and twenty-first years. She never had these attacks at any other period of her life. Recently she has had attacks, lasting a half-hour to an hour, in which the person at whom she was looking became indistinct and seemed to have only one eye. No other phenomena were noticed. A maternal aunt of the patient suffered from severe bilateral headache. The mother's family and the father of the patient were gouty, and the patient has indications of a gouty tendency. No history of epilepsy in the family could be obtained.

When four and a half years old A. lost her balance while sliding down the balusters, and fell a distance of about eight or nine feet. She was on her hands and knees, trying to get up, when her mother reached her, and seemed to be confused. She complained of intense pain in the back of the head and neck. A red spot was seen upon the right side of the head, where she struck the floor. In a few minutes after the fall she became unconscious and appeared to be dead. After a short time she began to scream: "Oh, mamma; I am falling!" and this screaming continued for more than two hours. Four hours after the fall she seemed much excited, could not sleep, repeated long stories, but did not complain of pain. The next day and for several successive days her mind was exceedingly active.

Some months after the fall the mother observed an attack in which the right arm fell powerless to the side of the body, and the whole right side seemed paretic for about half an hour. Headache also was complained of. These attacks of transitory paralysis occurred during many years, and some attacks seemed to be aborted by medicine. Sometimes these attacks were on the right side and sometimes on the left side, but they were never on both sides at the same time. As she grew older and became more able to explain her symptoms she complained of numbness and loss of sense of position in the paralyzed limbs, of paraphasia, and intense headache on the side opposite to the affected limbs when she had the attacks of paresis. The pain then was intense, and was in the motor region. The head pain was never on the same side as the weakness of the limbs, although the paresis and headache were first on one side and then on the other. When quite a small child she said she had five different kinds of headache. She was never entirely free from this pain. In her attacks the numbness would begin in the fingers and pass upward, and as it passed upward the part of the limb below nearly always lost all sensation. When the numbness reached the tongue she was unable to speak correctly; she could say words, but they conveyed no meaning. No visual symptoms existed at any time, except that dark spots and lines may have been seen many years ago during some of the attacks, although considerable doubt exists in regard to this. During the past year she complained of dimness of vision, but only in one attack. She is quite certain that she never had hemianopsia or diplopia.

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[1] After the above was written the patient informed me that he had had an attack of unconsciousness about a week ago while in a railroad train. A fellow-passenger told him that his eyes were drawn up and to the right, that he trembled slightly in all his extremities, and that his upper limbs became flexed and rigid for a short time. This account makes the diagnosis of epilepsy even more probable.

After she was eleven years of age the attacks became much less frequent, and between her fifteenth and eighteenth years they were very rare. No convulsions were ever observed, and as she was under constant observation, and convulsions were feared and watched for, it is probable that they would have been detected if they had occurred. The headache in the motor region sometimes occurred without the numbness of the opposite upper limb, but very seldom, and only when medicine had been administered; the numbness never occurred without the headache except on one occasion during the past year, when a peculiar dead feeling was experienced in both hands. This was the only time that paræsthesia existed in both hands at the same time. The headache, when in the frontal region, was sometimes attended by hallucination of smell; a sweet odor was perceived when none existed, and no numbness was felt at these times.

About three years ago she fell from a wagon and struck her left cheek, just below the eye. The attacks did not immediately become more frequent, but only after a year or two.

On October 4, 1899, about midday, an attack of numbness began in the right upper limb and extended to the face, and she lost the power of correct speech. She took a nap and awoke feeling well. The day before this attack occurred she had spoken of feeling unusually well. Toward night on October 4th she suddenly began to complain of severe pain in her head. She was placed on the floor by her mother. Her father, who is a physician, was called, and reached her a few minutes after the attack commenced. She was quiet, and he thought she was unconscious. She soon began to complain of her head again, and a very small amount of chloroform was administered. After the attack was over she had a very imperfect recollection of what had happened during the attack. Her mother said that in the beginning of the attack the head and eyes were drawn to the right, and she described the attack as a convulsive one. She was possibly excited, and may not have made careful observation.

After the fall from the carriage, about three years ago, a slight lateral curvature was noticed in the lumbar region, and the entire spine is now exceedingly sensitive to slight pressure, and much spontaneous pain is complained of in the back.

In my examination made October 13, 1899, the pupils were equal and reacted to light and in convergence. The patellar reflex was normal. No disturbances of objective sensation existed anywhere. No tender spots were found beneath the breasts, in the inguinal region, or on the head. The grasp of the hands was equal and good.

A urinary examination was made by Dr. S. M. Hamill, October 13, 1899: Reaction, normal; specific gravity, 1010; albumin, none; sugar, none; no pus-cells; no casts; normal epithelium.

Dr. Hamill made a careful examination of the thoracic and abdominal organs. There was no evidence of pulmonary tuberculosis or organic heart disease; a slight systolic whiff was heard just below the pulmonary area, and was believed to be functional, on account of its location and limitation, together with the normal pulmonary sound. The aortic sound was very much accentuated, as a result of high arterial tension. The abdominal examination revealed a freely movable right kidney and apparently a gastroptosis as well as an enteroptosis. The transverse colon was considerably dilated. The spleen and liver were not enlarged

These cases may be briefly summarized: The first patient, a man of fifty-one years, who had used alcohol since his eighth year and smoked to great excess, began, when about forty-four years old, to have numbness in the right side of the tongue and inability to speak; these attacks lasted a minute or two. The attacks occurred at first only once or twice a year, but are now frequent. When about forty-eight years old he began to have similar attacks of numbness in his right upper limb, and after a short time in his right lower limb, these attacks lasting a minute or two. The numbness of the hand was attended with weakness of the part. The paræsthesia has always been confined to the right side of the body, and has been associated with impaired function of the part affected, as shown by loss of speech and the weakness of the hand. These attacks may be regarded as petit mal. He does not lose consciousness in these attacks. The excessive use of alcohol and tobacco may have etiological importance, and may possibly account for the late development of the epilepsy. Erb[1] has stated very positively that excessive use of tobacco causes arterio-sclerosis. The development of the numbness followed a regular course: First the tongue was affected, then the right upper limb, and finally the right lower limb. The major attacks began after the attacks of numbness had existed for some time, and they were preceded by an aura; a feeling of unusually good health, followed by drowsiness. The bewilderment following an attack of unconsciousness, and the cry "Uh-uh-uh" preceding the attack, are suggestive of epilepsy.

The absence of convulsions is a noteworthy feature of the case; but, recognizing the case as one of epilepsy, we are prepared for the appearance of convulsions at any time. Paræsthesia is by no means unknown in epilepsy, either preceding or following a convulsive attack, and we need hardly be surprised that it should exist without the convulsions. The strict limitation of this paræsthesia to the right side of the body is a most interesting fact. The periods of unconsciousness and the paræsthesia beginning in the back of the right hand, when it heralds a major attack, are also noteworthy.

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[1] Deutsche Zeitschrift für Nervenheilkunde, vol. xiii., Nos. 1 and 2.

I have searched for some indication of word-blindness, of agraphia, of word-deafness, of motor aphasia—inasmuch as the symptoms point to involvement of the left cerebral hemisphere—of hysteria, but none are present. The absence of headache, of scintillating scotoma, makes a diagnosis of migraine doubtful. I should prefer to regard the case as a *forme fruste* of epilepsy—as an example of sensory epilepsy.

The second case is much more like one of migraine. Unilateral headache, with transitory paresis and paræsthesia on the opposite side of the body, with paraphasia, may be called migraine. The commencement of the attacks in early life, after a severe fall upon the head, must arrest attention. This injury seems to have had etiological importance. The mother of the child had peculiar attacks of headache, and a history of gout in the family was obtained. The possible relation of this case to epilepsy is shown by the occurrence of an attack of doubtful interpretation seventeen years after the attacks of paræsthesia and paresis began. The attacks of paresis would afford an interesting theme for some remarks, but paralysis as an epileptic equivalent has been recently ably discussed before this Society by McConnell,[1] and I merely refer to the fact that in my case the attacks were of long duration—a half-hour or so.

We have, therefore, two cases in which attacks of unilateral paræsthesia and paresis occur, associated with disturbance of speech; and in one case with periods of unconsciousness; in the other with severe headache on the opposite side. The headache is a very important symptom, but no less important are transitory paresis, paræsthesia, and disturbance of speech. Paderstein[2] says that hemicrania is the most common symptom of ordinary migraine, but that, according to most authors, the statement "no migraine without hemicrania" is incorrect.

Cases of sensory epilepsy are described in the literature in connection with migraine. Féré, for example, says that epilepsy is among the frequent accompaniments of ophthalmic migraine, especially the partial epilepsy. Ophthalmic migraine may develop in a person who has previously had partial epilepsy and still has attacks of paræsthesia in the side of the body in which the epileptic movements occurred. He gives a good example of this. He speaks also of other forms—of cases in which the migraine may precede the epilepsy, of cases in which the two conditions occur together, and of cases in which the attacks of migraine become less severe after the appearance of partial epilepsy. The cases reported by Féré[1] are very instructive.

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[1] Journal of Nervous and Mental Disease, June, 1899, p. 355.

[2] Paderstein. Deutsche Zeitschrift für Nervenheilkunde, vol. xv., Nos. 5 and 6, p. 332.

Charcot also was much interested in ophthalmic migraine, and a case published by him is well worthy of attention. A man had attacks of a peculiar character, which began generally about six o'clock in the evening with right lateral hemiopia and scintillating scotoma. This period lasted about an hour; the hemiopia and scotoma disappeared, and were followed by left-sided headache and aphasia for all forms of speech—*i. e.*, word-deafness, word-blindness, motor aphasia, and agraphia. The second period also lasted about an hour, and then paræsthesia of the right hand and upper and lower lips developed; the headache increased and nausea became pronounced; finally, the patient fell asleep, and the attack was over. No history of convulsions is given. Charcot[2] remarked that paræsthesia of the limbs and face and aphasia very rarely occur in migraine; but he also stated that ophthalmic migraine may be associated with transitory hemiplegia, epileptoid attacks, and other phenomena.

The severe forms of ophthalmic migraine may be regarded as "associated with" epilepsy, if one so pleases; but when epilepsy occurs with migraine it is well to recognize that there is probably some connection between the two diseases, and that it is not merely a chance occurrence of two diseases in the same person. The important fact is that a person who has ophthalmic migraine, with aphasia and unilateral paræsthesia, may some day develop Jacksonian convulsive attacks. Whether this is to be regarded as epilepsy associated with migraine, or the whole is to be considered an epileptic manifestation, can be determined only after we understand better the pathology of the two diseases. It may be that ophthalmic migraine and epilepsy have the same cause in some cases; it may be that the migraine changes the structure of the cerebral vessels and of the brain and renders the latter liable to the epileptic disease. In my first case no history of headache or visual symptoms could be obtained; it would seem improper to regard these attacks of paræsthesia and paresis from which the patient suffered as a *forme fruste* of ophthalmic migraine. It seems better to look upon them as a *forme fruste* of epilepsy.

Diller[3] presented to this Society in 1895 a case which, in many of its features, closely resembles my first case. He described it as a case of sensory epilepsy. The patient, a man, twenty-five years old, was kicked by a mule on the right side of the forehead, and was unable to work for about a week. About a year and a half later he had his first attack. He experienced a feeling of numbness and tingling in the left toes—a sensation he described as "pins and needles." This paræsthesia slowly ascended the leg, thigh, and trunk, went down the left arm to the fingers, then up the left side of the neck and face, and involved the left side of the tongue. When the paræsthesia had reached the middle of the trunk in its upward march it began to fade from the toes, and precisely as it had come it passed away. An absolute loss of muscular power passed over and faded from the left side, just as the paræsthesia had done. The attack lasted from twenty to thirty minutes. He was entirely conscious during the attack. Subsequent attacks were similar, and nearly half of them began in the left fingers. When about twenty-eight years old he had a general convulsion, which began with the manifestations of the sensory attacks, and in this attack he lost consciousness. These sensory attacks lasted about a year and a half before a convulsive attack occurred.

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[1] Les epilepsies et les epileptiques, p. 50.

[2] Nouvelle iconographie de la Salpêtrière, 1895, No. 1, p. 3.

[3] Journal of Nervous and Mental Disease, 1895, p. 284.

Diller thought that the theory of an irritated sensory cortex was hardly acceptable, because the paræsthesia did not always begin in the same part, although he stated that a brain tumor might have been present.

Möbius speaks of the difficulty attending the diagnosis of epileptic migraine. He refers to the fact that Féré regarded epilepsy and migraine as expressions of the same disease, and quotes this sentence from Féré's writings: "Among the phenomena accompanying partial epilepsy or alternating with it, it is necessary to mention sensorial migraine, and especially ophthalmic migraine, which may serve as a type, and which, when isolated, constitutes a veritable sensorial epilepsy, with phenomena of exhaustion, hemianopsia, and sometimes somnolence." Möbius goes on to say that from this stand-point there can be no difference between migraine and epilepsy, and he thinks Féré makes a mistake in basing his opinion merely on the symptoms. Migraine, according to Möbius, is an hereditary disease, unaltered in its manifestations by transmission—which, however, is not always true. It usually remains without change during the life of the patient; it does not cause mental enfeeblement, and, therefore, it is different from epilepsy. He acknowledges, however, that in certain cases it may be impossible to say whether epilepsy or migraine is present; but when convulsions appear the disease is to be regarded as epilepsy with migrainoid symptoms. The symptoms of migraine are of a sensory character, and he would not diagnosticate a case as one of migraine when convulsive movements are present, even though they are as slight as the drawing of the mouth and tongue to one side. The phenomena accompanying the convulsions are to be regarded as simulating migraine, but the case is one of larvated epilepsy. Möbius quotes three cases reported by Féré as cases of ophthalmic migraine, and states his (Möbius[1]) opinion that they should be classed under epilepsy. This is an instance of how two distinguished neurologists may disagree in the names to be employed in diagnosticating between epilepsy and migraine. Möbius also says that if a patient with migraine has attacks of unconsciousness the suspicion of epilepsy may be entertained. Surely, attacks of unconsciousness without migraine, as in my first case, are no less suspicious of epilepsy. The difficulties in making a distinction between certain forms of epilepsy and migraine are clearly presented in Möbius' words, and his work is one of the best that have appeared on the subject of migraine.

The heredity in migraine is usually direct, but it may occasionally be indirect. Gowers[2] refers to a woman, aged forty-six years, who had migraine for many years (paroxysmal headaches, with dimness of sight and vomiting); one of her children was epileptic, and her mother had been epileptic and insane. He had also a brother and sister as patients; one suffered from epilepsy, the other from migraine. Gowers speaks of the relation between migraine and epilepsy, and says he has seen cases in which epilepsy succeeded migraine, and the epileptic fits seemed, as it were, to grow out of the attacks of migraine.[3]

Oppenheim[4] observed a case in which attacks of hemiparesis intercurred with the migraine attacks; in my second case the hemiparesis and the hemicrania occurred simultaneously.

In view of the abortive forms of epilepsy which occasionally appear, it may be well to repeat the definition of epilepsy given by one of the most recent writers on this subject—Binswanger,[5] in the Nothnagel series of monographs. "Epilepsy," he says, "is a very chronic disease of the central nervous system, and is produced by the most different causes. Its clinical phenomena exist in repeatedly occurring convulsive attacks, with unconsciousness, or in partial manifestations of these attacks, or in psycopathic attending or following phenomena."

A still more recent definition is given by Donath.[6] According to him, epilepsy is a morbid excitement of the cerebral cortex, increasing suddenly, returning periodically, having a typical course, and diminishing rapidly. Whether the attacks occur with unconsciousness and amnesia or not depends on the severity and extent of the irritation. Unconsciousness and amnesia are not a necessary part of the attack.

It is not my intention to give the opinions of all the writers regarding the relation of epilepsy to migraine; they may be found in the various text-books. A proper view of the subject seems to me to be the following:

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[1] Die Migräne, p. 76, Nothnagel's Specielle Pathologie und Therapie.

[2] A Manual of Diseases of the Nervous System, vol. ii., second English edition.

[3] Loc. cit., p. 849.

[4] Lehrbuch des Nervenkrankheiten, first edition, p. 720.

[5] Die Epilepsie. Nothnagel's Specielle Pathologie und Therapie.

[6] Donath. Archiv für Psychiatrie, vol. xxxii., No. 2, p. 339.

1. Attacks of migraine occur associated with nausea and vomiting; this form is known as simple migraine, and usually remains unaltered during the life of the patient.
2. In other cases visual disturbances (hemianopsia, scintillating scotoma, amaurosis, etc.) are associated with the migraine, and the disease is then known as ophthalmic migraine.
3. When paralysis of the ocular muscles occurs with the migraine the disease is described as ophthalmoplegic migraine.
4. Migraine, especially the ophthalmic form, is related to epilepsy, and the attacks of migraine may precede for many years the convulsive attacks of epilepsy, although in most cases of migraine no convulsions are ever detected.
5. In some cases epilepsy appears in the form of one or more of the disturbances seen occasionally with migraine, and later, even after many years, convulsions develop. The disease may be epilepsy from the beginning. It matters little, with our uncertain knowledge of the pathology of the two diseases, whether we regard these as abortive cases (*formes frustes*) of migraine that later become associated with epilepsy, or as abortive forms of epilepsy (sensory epilepsy), in which the convulsions later become apparent, provided we recognize a relation between some forms of migraine and epilepsy.

FACIAL SPASM AND ITS RELATION TO ERRORS OF REFRACTION.[1]

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Under the general designations "facial spasm," "mimic spasm," "habit spasm," "histrionic spasm," "painless tic," "convulsive tic," and "palmus" are included several groups of cases presenting the common clinical feature of spasm of the muscles supplied by the seventh or facial nerve.

The following groups of cases are recognized by Osler:[2]

1. The secondary form following paralysis, and consisting in spontaneous clonic twitchings recurring at irregular intervals in the paralyzed muscles.
2. Cases due to the irritation of an organic brain lesion either of the cortex or of pressure on the facial nerve from an aneurism or tumor.
3. In many cases no cause can be assigned—the idiopathic form of Gowers.

[1] Read before the Colorado State Medical Society, June 20, 1899.

[2] American Text-book of the Theory and Practice of Medicine, vol. I. p. 832.